Case Report
JCAM

Biventricular thrombi and pulmonary embolism in a young woman with peripartum cardiomyopathy

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Abstract

Postpartum cardiomyopathy (PPCM) is a rare form of left ventricular (LV) systolic dysfunction of unknown etiology that occurs in previously healthy women in the final month of pregnancy or postpartum. It is characterized by LV systolic dysfunction and congestive heart failure and is associated with thromboembolic events and LV thrombus. Here we presented a postpartum cardiomyopathy patient with biventricular thrombosis and pulmonary thromboembolism.

Keywords

Biventricular Thrombus; Pulmonary Embolism; Postpartum Cardiomyopathy

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Introduction

Postpartum cardiomyopathy (PPCM) is a rare form of left ventricular (LV) systolic dysfunction of unknown etiology that occurs in previously healthy women in the final month of pregnancy or postpartum [1]. It can occur in the third trimester of pregnancy or in the first month after delivery [2]. It is characterized by LV systolic dysfunction and congestive heart failure and is associated with thromboembolic events and LV thrombus [3]. LV apical thrombus is seen more commonly in patients with PPCM. Biventricular thrombus formation is rarely seen in PPCM. Cerebrovascular events, peripheral embolism, and pulmonary embolism can be seen in patients with PPCM due to the cardiac thrombus. Thrombolytic agents can be distinctively used in patients with PPCM. We present a patient with PPCM who was admitted to the intensive care unit with biventricular thrombus and pulmonary embolism.

Case Report

A 22-year-old woman presented to the emergency department with a sudden onset of dyspnea and palpitation 15 days after delivery. Her medical history showed that she had 3 healthy children and was discharged on the postoperative day 2 after an uncomplicated pregnancy and cesarean section. She had not been taking any medication. Physical examination revealed a blood pressure of 90/60 mmHg, heart rate of 115/min, and respiratory rate of 28/min. The auscultation of the heart revealed a grade 2/6 pansystolic murmur. Her oxygen saturation was 88%. The remainder of the examination was unremarkable. Chest X-ray showed the enlargement of the cardiac silhouette (the cardiothoracic ratio was 70%) and pulmonary vascular congestion. Electrocardiogram revealed sinus tachycardia and right axis deviation. Transthoracic echocardiogram revealed reduced left and right ejection fraction (EF) with increased left end-diastolic ventricular diameter (62 mm), mild mitral regurgitation, bilateral ventricular apical thrombus due to the severe global hypokinesia (Left EF 30%, right EF 35%). The fresh thrombus in the right ventricle was attached to the septum and mobile whereas the two oval-shaped thrombi in the LV were immobile (Figure 1-2). Because the physical examination and

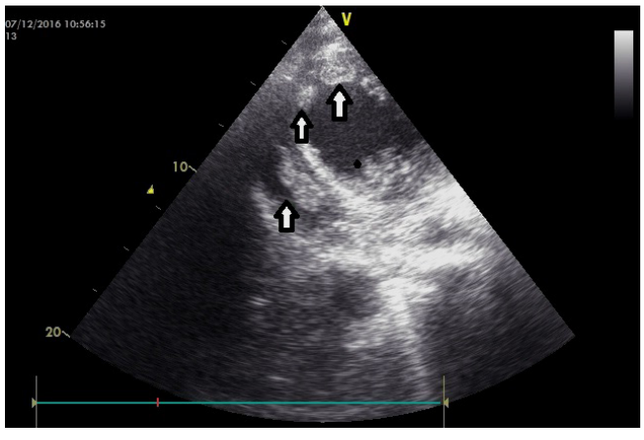

Figure 1. Biventricular apical and septal thrombi

echocardiographic findings suggested the possibility of pulmonary embolism, computed tomography angiogram was ordered and was consistent with pulmonary embolism. (Figure3). Laboratory studies showed an elevated level of d-dimer and

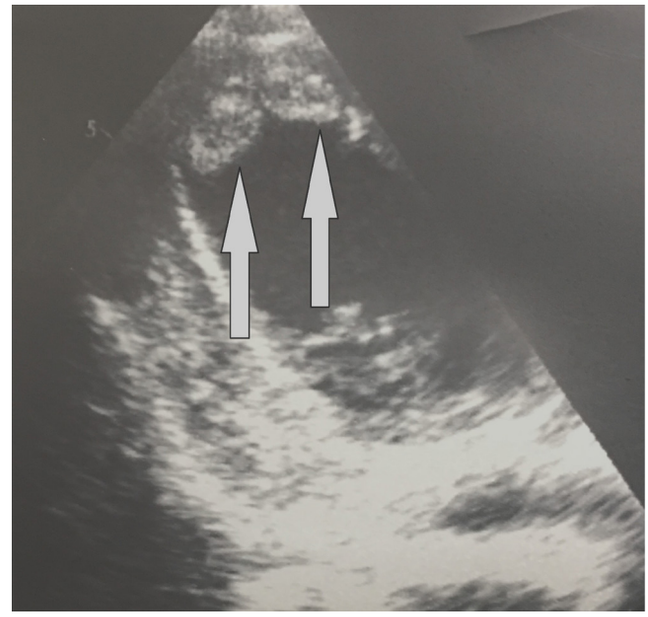

Figure 2. Left ventricular apical thrombi

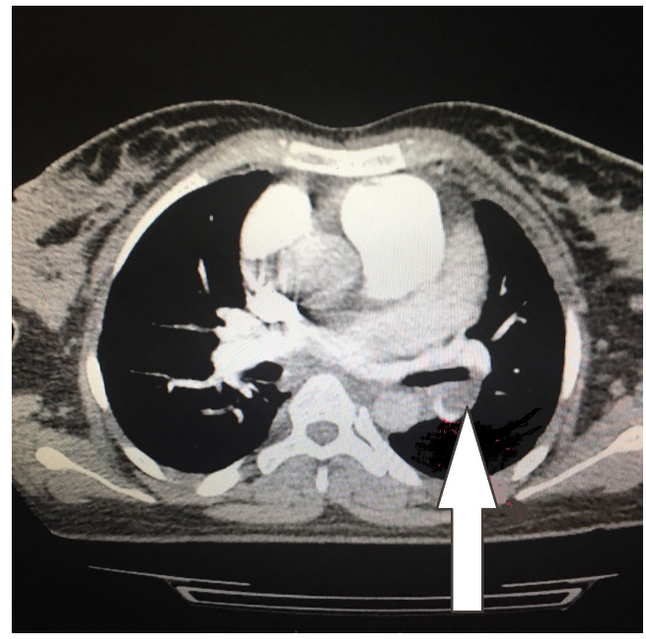

Figure 3. Pulmonary embolism

troponin I whereas protein C/S, anticardiolipin antibody, and antithrombin III levels were within normal limits. Bromocriptine was added to the conventional heart failure therapy. She was started on intravenous unfractionated heparin (target activated partial thromboplastin time 50–70 s). Thrombolytic therapy was reserved in case of hemodynamic instability. Her dyspnea and tachycardia resolved and her blood pressure went up to the normal levels during follow-up. The control echocardiogram revealed that the fresh thrombus in the right ventricle had disappeared while the thrombi in the left remained the same. She was started on oral warfarin therapy and was discharged after achieving an international normalized ratio (INR) of 2-3.

Discussion

Cardiomyopathy during the pregnancy occurring in previously healthy women who were in the last month of pregnancy or within five months of delivery was firstly described by Demakiset al. in 1971. LV EF of 45% or less was considered as a

systolic dysfunction [1-2]. The exact incidence of PPCM is unknown, and there is a significant difference between northern and southern hemisphere. The incidence is 1 in 300 in Haiti, 1 in 1000 in Africa, 1 in 3000 in the USA and Europe [1]. Even though PPCM has a high morbidity and mortality rate, the pathogenesis of PPCM is still unknown. The risk factors include black race, advanced age, multifetal pregnancy, prolonged use of beta blocker, history of hypertension and complicated pregnancy [4]. Viral myocarditis, abnormal response to the hemodynamic stress of pregnancy, immune-mediated injury, and selenium deficiency were considered as etiologic factors, but none of them can be proven [5]. The "oxidativestress–prolactinaxis" hypothesis underlines the role of prolactin in the pathogenesis of PPCM. Oxidative stress triggers the activation of a lysosomal enzyme, cathepsin D, which in turn cleaves serum prolactin into it antiangiogenic and proapoptotic 16-kDa prolactin subfragment. Prolactin inhibition is being explored as a novel PPCM treatment. The coagulation and fibrinolytic systems undergo significant change during the pregnancy. Von Willebrand factor, factor VIII, fibrinogen, plasminogen activator inhibitor 1-2 levels and platelet aggregation are increased, and protein C and S levels are decreased during the pregnancy. All of these changes result in a hypercoagulable state [6]. In addition to hypercoagulable state, LV dilatation and systolic dysfunction are some of the predisposing factors to thrombus formation in the cardiac structures [3, 6]. LV apical thrombus is more commonly seen in PPCM. However, bilateral apical thrombus formation is rarely seen [3]. The cardiac thrombus formation may cause thromboembolic complications such as myocardial infarction, pulmonary embolism, stroke, and ischemia of extremities.

We presented the co-occurrence of biventricular apical thrombus and pulmonary embolism. The treatment of the ventricular thrombus is still controversial. Some case series showed that unfractionated heparin could be effective on fresh thrombi and dissolve the thrombi [7]. Kim DY et al. reported that the anticoagulation therapy was successfully used to treat the biventricular thrombus without any embolic complications [8]. This treatment was supported in another article published by Nishi I et al. [7]. There is no consensus on the treatment of embolic complications of biventricular thrombus. Koc M et al. reported the successful thrombolytic therapy used in a patient with biventricular thrombus and cerebral embolism [9]. Yaméogo NV et al. reported the successful streptokinase therapy in a patient with biventricular thrombus, pulmonary embolism, and acute limb ischemia [10]. We did not use the fibrinolytic therapy since the patient had not developed hemodynamic instability. We used conventional heart failure medications with bromocriptine and unfractionated heparin followed by warfarin. The fresh thrombus in the right ventricle disappeared during follow-up while the thrombi in the LV remained in the same location. The patient was hemodynamically stable during follow-up. She was discharged with oral warfarin therapy (target INR 2-3).

In conclusion, the anticoagulant therapy can be given without thrombolytic therapy to treat biventricular thrombi in PPCM patients with active bleeding or high risk of bleeding. The risk-benefit ratio of thrombolysis treatment must be carefully evaluated.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

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